Management of Peripapillary Choroidal Neovascular Membrane in Patients With Idiopathic Intracranial Hypertension

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Abstract: OBJECTIVE To report the clinical features and treatment outcomes of patients with peripapillary choroidal neovascular membrane (CNVM) secondary to idiopathic intracranial hypertension (IIH). METHODS Retrospective, multicenter chart review of patients diagnosed with peripapillary CNVM in the course of the treatment and follow-up of IIH. RESULTS Records were reviewed from 7 different institutions between 2006 and 2016. Ten patients (13 eyes) with a diagnosis of IIH and at least 3 months of follow-up developed CNVM. Three of the total 10 patients developed bilateral CNVM. The mean time from the diagnosis of IIH to CNVM diagnosis was 41 months. Mean follow-up period was 8 months after diagnosis of CNVM. All patients were treated with acetazolamide for IIH. Seven eyes were observed, and 6 eyes were given anti-vascular endothelial growth factor (anti-VEGF) injections, including bevacizumab, ranibizumab, and aflibercept. All CNVMs regressed with subretinal fibrosis, and visual acuity improved in most patients. Papilledema resolved in only 1 eye, while the other 12 eyes had persistent papilledema at last follow-up. CONCLUSIONS Peripapillary CNVM, a rare complication of IIH, often resolves spontaneously with treatment of IIH. In vision-threatening and/or persistent cases, intravitreal anti-VEGF treatment may be a safe and effective therapeutic option.

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Objective: To report the clinical features and treatment outcomes of patients with peripapillary choroidal neovascular membrane (CNVM) secondary to idiopathic intracranial hypertension (IIH).

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Conclusions: Peripapillary CNVM, a rare complication of IIH, often resolves spontaneously with treatment of IIH. In vision-threatening and/or persistent cases, intravitreal anti-VEGF treatment may be a safe and effective therapeutic option.

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Peripapillary choroidal neovascular membrane (CNVM) is an infrequent cause of vision loss in patients with idiopathic intracranial hypertension (IIH) (1–3). The prevalence of peripapillary CNVM in IIH has previously been reported as 0.53% (4). There are few reports on the management of IIH-associated peripapillary CNVM. The diagnosis of CNVM is made clinically based on the identification of a deep peripapillary hemorrhage and a grayish-white opacity merged within the area of optic disc edema. This grayish-white opacity indicates the presence of a membrane, which becomes increasingly more apparent on ophthalmoscopy as intracranial pressure is reduced and papilledema resolves. Diagnosis is assisted by subretinal fluid demonstrated on optical coherence tomography (OCT). Likewise, fluorescein angiography (FA) demonstrates hyperfluorescence secondary to CNVM leakage, along with an area of hypofluorescence as subretinal hemorrhage blocks choroidal transmission.

In most cases, medical or surgical treatment aimed at lowering the intracranial and/or optic nerve sheath pressure results in progressive involution of peripapillary CNVM (20–23). For persistent or symptomatic CNVM, intravitreal anti–vascular endothelial growth factor (anti-VEGF) injection is the treatment of choice (4–7). We describe the

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The authors report no conflicts of interest.

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TABLE 1. Demographics and clinical features of patients with IIH and CNVM

	(n = 10 Patients/13 Eyes)
Age	35 (15–54) years
Sex (male/female)	8/2
BMI	35 (19–48) kg/m^2
MRI/MRV	Normal
LP opening pressure	>30 cm H_2O
Subretinal hemorrhage and/or fluid	11 eyes
Time for CNVM formation	41 (2–112) months
Follow-up time after CNVM	8 (3–54) months
Treatment for IIH	Oral acetazolamide

Data are expressed as the mean (range) of cases.
BMI, body mass index; CNVM, choroidal neovascular membrane; IIH, idiopathic intracranial hypertension; LP, lumbar puncture; n, number; MRV, magnetic resonance venography.

management of 10 patients (13 eyes) who developed peripapillary CNVM in association with IIH.

METHODS

Research conducted was in compliance with the Health Insurance Portability and Accountability Act and Declaration of Helsinki, while abiding to all regional, national, and international laws of the institutions involved in this study. Every effort was made by the investigators to protect the rights of the patients. In addition, this study followed the tenets of the ethics committee of each contributing center and was approved by the institutional review board of the University of Michigan.

Patients evaluated between January 1, 2006, and December 31, 2016, were reviewed and selected from the following institutions: University of Michigan, W.K. Kellogg Eye Center, Ann Arbor, Michigan; Department of Ophthalmology and Visual Neurosciences, University of Minnesota, Minneapolis, Minnesota; Department of Ophthalmology, Duke University Eye Center, Durham, North Carolina; Department of Ophthalmology, University Hospital Zurich and University of Zurich, Zurich, Switzerland; Department of Ophthalmology, Washington University, St. Louis, Missouri; Department of Ophthalmology, Indiana University School of Medicine, Indianapolis, Indiana; and Department of Ophthalmology, Hôpital Delafontaine, Saint-Denis, France. A diagnosis of IIH and a minimum of 3 months of follow-up after CNVM treatment were required for study inclusion. Collected data included age, sex, body mass index (BMI), fundus examination findings, best-corrected visual acuity (BCVA) at the time of CNVM diagnosis and at last follow-up, visual field mean deviation (VFMD), treatment for IIH and CNVM (if any), follow-up time, MRI and magnetic resonance venography (MRV) results, lumbar puncture (LP) opening pressures, and status of papilledema and CNVM at the last follow-up. Patients with a diagnosis of CNVM before IIH diagnosis were excluded.

Statistical Package for Social Science v 22.0 software (SPSS, Chicago, IL) was used to conduct the statistical analyses. Comparisons between the visits were made using Wilcoxon signed-rank. All the reported *P* values were 2-tailed, and those less than 0.05 were considered to be statistically significant.

RESULTS

Based on data collected over a 10-year period from one of the study sites (University of Michigan, W.K. Kellogg Eye Center), we report a 0.96% prevalence of CNVM in patients with IIH-associated papilledema and at least 3 months of documented follow-up.

Patient characteristics and treatment results are summarized in Table 1. A total of 13 eyes of 10 patients (8 women and 2 men) met the inclusion criteria, with a mean age of 35 years (range 15–54 years). The mean BMI was 35 kg/m^2 (range: 19–48 kg/m^2). Brain MRI and MRV were normal except for dilated optic nerve sheaths in all patients. LP opening pressures were over 30 cm H_2O in all patients. All 13 eyes with CNVM also had papilledema. Peripapillary subretinal hemorrhage and subretinal fluid were found in 10 eyes, while 1 eye only had peripapillary subretinal fluid in association with CNVM (Fig. 1). FA performed in 4 of the 10 patients included in this study demonstrated hyperfluorescence in the midphase with late leakage, indicative of a peripapillary CNVM. The mean elapsed time between diagnosis of IIH and documentation of a diagnosis of CNVM was 41 months. The mean length of follow-up after CNVM diagnosis was 8 months (range: 3–54 months). All patients with IIH were treated with oral acetazolamide. Symptomatic or vision-threatening CNVMs were treated with intravitreal anti-VEGF injections in 6 eyes. Anti-VEGF injections were given at the discretion of the treating ophthalmologist guided by OCT findings. Three eyes received 1 bevacizumab (1.25 mg/0.05 mL) (Avastin; Genentech, Inc, South San Francisco, CA) injection, 1 eye received 3 bevacizumab injections, 1 eye received 2 bevacizumab and 4 aflibercept (2.0 mg/0.05 mL) (Eylea; Regeneron, Tarrytown, NY) injections, and 1 eye received 2 ranibizumab (0.5 mg/0.05 mL)

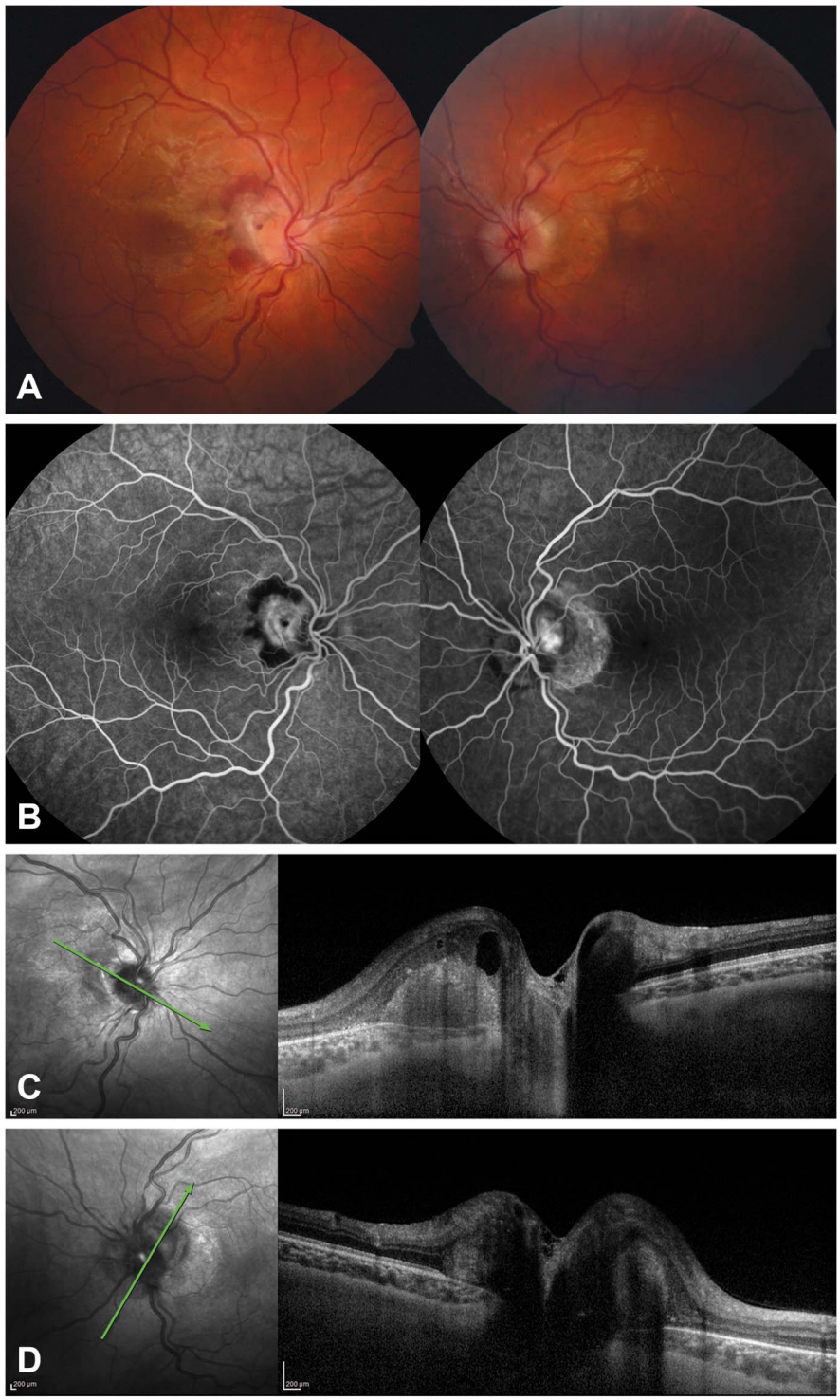

FIG. 1. Patient 4. **A.** There is papilledema with peripapillary subretinal hemorrhage and fluid in the right eye and papilledema with peripapillary atrophy in the left eye. **B.** Midphase fluorescein angiogram reveals peripapillary hyperfluorescence surrounded by a ring of hypofluorescence in the right eye. In the left eye, there is peripapillary hyperfluorescence due to CNVM and an adjacent crescent-shaped hyperfluorescent window defect. **C.** OCT of the right eye shows intraretinal and subretinal fluid consistent with an active CNVM. **D.** OCT of the left eye demonstrates peripapillary thickening with outer retinal atrophy. Green arrows indicate the direction of the OCT imaging scan. OCT; optical coherence tomography; CNVM, choroidal neovascular membrane.

TABLE 2. Demographic, clinical, and treatment features of patients with IIH and CNVM

Pt	Sex	Age (yrs)	Laterality	Imaging Modality Used in Diagnosis	Initial VA	VA at the Last Visit	Treatment	Follow-up Time (Months)	Last Status CNVM	Last Status Papilledema
1	F	32	Left	FA	20/20	20/60	Bevacizumab × 3	56	Resolved	Improved
2	F	26	Right	OCT	20/500	20/200	Ranibizumab × 2	8	Resolved	Improved
3	M	21	Right	FA + OCT	20/30	20/40	Observation	6	Resolved	Improved
4	F	51	Right	Fundus photography	20/20	20/20	Observation	8	Resolved	Improved
			Left	Fundus photography	20/25	20/25	Observation	8	Resolved	Improved
5	F	15	Right	OCT	20/40	20/40	Observation	5	Resolved	Resolved
6	F	54	Right	FA + OCT	CF	20/250	Bevacizumab × 1	21	Resolved	Improved
7	F	45	Right	OCT	20/50	20/40	Observation	8	Resolved	Improved
			Left	OCT	Hand motion	20/100	Bevacizumab × 2, aflibercept × 4	8	Resolved	Improved
8	F	23	Right	Fundus photography	20/250	20/250	Bevacizumab × 1	18	Resolved	Improved
9	M	48	Left	OCT	20/50	20/200	Bevacizumab × 1	3	Resolved	Improved
10	F	30	Right	FA	20/40	20/32	Observation	14	Resolved	Persistent
			Left	FA	20/400	20/400	Observation	14	Resolved	Persistent

CNVM, choroidal neovascular membrane; F, female; FA, fluorescein angiography; IIH, idiopathic intracranial hypertension; M, male; OCT, optical coherence tomography; Pt, patient; VA, visual acuity.

(Lucentis; Genentech, Inc) injections. Seven eyes with non–vision-threatening CNVMs were observed (Table 2). In eyes managed with observation, the mean interval from time of diagnosis to time of resolution of CNVM was 248 days (range: 132–435 days). The resolution of CNVM correlated with improvement of papilledema in these eyes, although persistent disc edema and elevation were noted at most recent follow-up in all eyes except one.

Mean BCVA at the time of CNVM diagnosis and at the last visit was 0.80 ± 0.91 logarithm of the minimum angle of resolution (logMAR) and 0.60 ± 0.44 logMAR, respectively ($P = 0.62$). At the end of the follow-up period, most untreated and treated eyes either had stable or improved BCVA (Table 2). Mean initial and last BCVA in the observed group was 0.32 ± 0.42 logMAR and 0.37 ± 0.40 logMAR, respectively ($P = 0.63$). Mean initial and last BCVA in the treated group was 1.58 ± 0.98 logMAR and 0.98 ± 0.16 logMAR, respectively ($P = 0.37$). Mean improvement in VA was 0.05 ± 0.19 logMAR in the observed group and −0.60 ± 1.10 logMAR in the treated group ($P = 0.258$).

Eight of the 13 eyes included in this investigation underwent automated (Humphrey) visual field testing at time of CNVM diagnosis and at most recent follow-up, which included 3 patients in the treatment group and 5 in the observation group. The average VFMD for both treated and untreated eyes at time of CNVM diagnosis and at most recent follow-up were −8.59 ± 5.60 dB and −5.91 ± 5.45 dB, respectively ($P = 0.19$). Mean initial and last VFMD in the observed group was −13.38 ± 7.08 dB and −8.43 ± 6.07 dB, respectively ($P = 0.50$). Mean initial and last BCVA in the treated group was 1.58 ± 0.98 logMAR and 0.98 ± 0.16 logMAR, respectively ($P = 0.37$). Mean change in BCVA was 01.82 ± 4.20 logMAR in the observed group and 2.68 ± 2.37 logMAR ($P = 0.54$) in the treatment group.

All CNVMs regressed with residual peripapillary subretinal fibrosis either after treatment (Fig. 2) or observation (Fig. 3). Papilledema resolved completely in only 1 eye, while the other 12 eyes had improved but persistent disc edema and elevation at last follow-up.

DISCUSSION

In our multicenter retrospective chart review of patients who developed peripapillary CNVM secondary to IIH, 10 patients (13 eyes) from 7 different institutions were included, spanning a 10-year period from the beginning of the anti-VEGF era in 2006. All patients with IIH were treated with acetazolamide. Six eyes were treated with intravitreal anti-VEGF injections, including bevacizumab, ranibizumab, and aflibercept. The remaining 7 eyes were managed with close follow-up. Regression of peripapillary CNVM was documented in all eyes.

Peripapillary CNVM is an infrequent complication of chronic papilledema and can cause significant vision loss in patients with IIH. Data on this rare disorder are limited to few case reports and small case series (8–14). The diagnosis of CNVM is based on a combination of clinical examination findings and features observed on OCT and FA. There is deep peripapillary hemorrhage associated with subretinal fluid detected on OCT. Although fluid around the disc is common in patients with IIH, the presence of a grayish-white opacity within this area of edema indicates the presence of a CNVM. On FA, there is hyperfluorescence in the area of leakage from the CNVM and hypofluorescence in the area where subretinal hemorrhage is present.

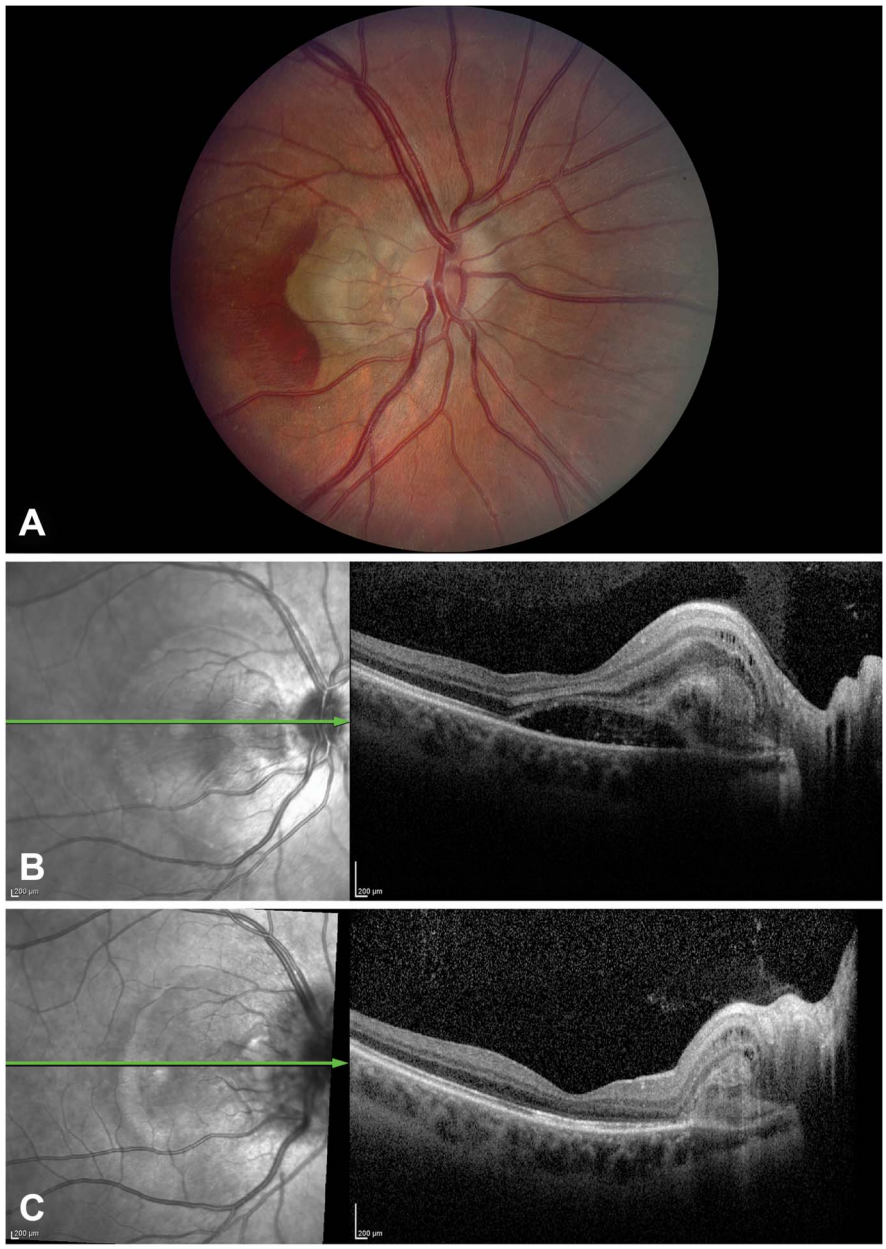

FIG. 2. Patient 2. **A.** The right fundus shows papilledema with peripapillary subretinal hemorrhage and intraretinal and subretinal fluid due to CNVM. **B.** Pretreatment OCT shows subretinal fluid extending into the macula. **C.** OCT shows resolution of subretinal fluid after treatment with 2 intravitreal ranibizumab injections. Green arrows indicate the direction of the OCT imaging scan. CNVM, choroidal neovascular membrane; OCT, optical coherence tomography.

Wendel et al (4) reported the largest series of peripapillary CNVM in patients with IIH-associated papilledema, before the development of VEGF inhibitors. The authors retrospectively reviewed 1,140 patients with IIH and identified 6 cases with peripapillary CNVM, for a prevalence of 0.53%. There were 5 women and 1 man with a mean age of 46.3 years. All patients had unilateral peripapillary CNVM and were treated with acetazolamide or furosemide to lower the intracranial pressure. They elected observation for 3 patients while 2 were treated with argon laser photocoagulation and 1 with photodynamic therapy. At the endpoint of their study, 3 of 6 CNVMs had regressed, 2 were smaller and inactive, and 1 was smaller but with a limited area of presumed activity. No recurrence of CNVM was noted.

In our study, we elected observation in 7 eyes, and intravitreal anti-VEGF injections were administered in 6 eyes. The decision to treat was based on the identification of a CNVM with persistent subretinal fluid threatening the fovea, expanding retinal exudation, increasing deep peripapillary hemorrhage, or a decline in VA out of proportion with the amount of optic disc edema. All but one eye in our study showed either stable or improved vision at the end of follow-up, and all CNVMs completely regressed.

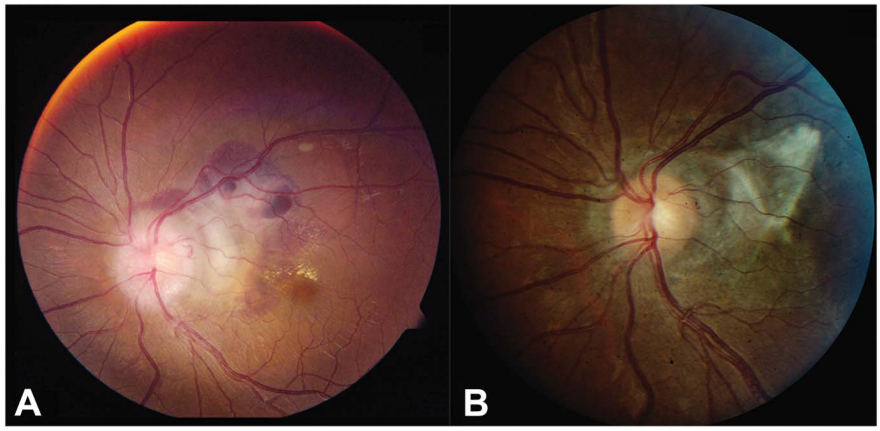

FIG. 3. Patient 10. **A.** Peripapillary CNVM and papilledema are present in the left eye. **B.** After 8 months of observation, there is regression of CNVM with subretinal fibrosis and diminished optic disc edema. CNVM, choroidal neovascular membrane.

The causal relationship between developing a peripapillary CNVM and papilledema is controversial. Kaeser and Borruat (15) reported a patient with bilateral papilledema and unilateral peripapillary CNVM with spontaneous involution of the membrane along with improvement of the patient's vision from 20/200 to 20/30 after treatment of IIH with acetazolamide for 1 year. However, Sathornsumetee et al (2) described a patient with IIH whose peripapillary CNVM did not regress and VA that did not improve after optic nerve sheath fenestration, despite dramatic improvement in the degree of papilledema. In our study, all 13 peripapillary CNVMs regressed with concurrent improvement in papilledema. It is unclear whether our findings are related to medical therapy of IIH, treatment of the CNVM, or simply due to the natural course of the CNVM.

The pathogenesis of peripapillary CNVM in patients with papilledema due to IIH is unclear, but it seems to be different from CNVMs that occur in other posterior segment diseases such as exudative age-related macular degeneration (16–19). In macular degeneration, retinal pigment epithelium dysfunction and Bruch membrane breaks occur as a result of retinal drusen deposition and inflammation, which subsequently promotes hypoxia and angiogenesis (19,20). Peripapillary CNVM in IIH might result from a pressure deformity of the Bruch membrane at the level of the optic nerve head (16). This combined with persistent hypoxic environment created by axonal swelling may initiate the angiogenesis cascade, leading to the neovascular membrane formation (21,22).

Management options for peripapillary CNVM in patients with IIH include observation, subretinal surgery, laser photocoagulation, photodynamic therapy, or intravitreal injection of anti-VEGF agents (3,6,16,23,24). Although therapeutic decisions must be made on a case-by-case basis, the use of anti-VEGF agents currently is the method of choice (1,7,23,24).

Our study has several limitations, including the method of diagnosis of CNVM. In each case, the diagnosis of CNVM was made clinically with supporting features observed on retinal imaging. Not all patients underwent both OCT and FA. Other limitations of our report include retrospective design and small sample size.

In conclusion, peripapillary CNVM in association with papilledema due to IIH may regress spontaneously with no vision loss with adequate medical treatment of IIH. In vision-threatening cases, intravitreal injection of bevacizumab, ranibizumab, or aflibercept may be an effective treatment option to preserve visual function.

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